

seems justifiable to be giving isoniazid to an adult suspected of being infected with *M. tuberculosis* organisms (no evidence of clinically active disease) if it is known or if the probability is strong that he was infected many years previously. In both cases, to place any reliance on isoniazid materially protecting such individuals runs contrary to what is presently known about the mode of action of isoniazid.

Possibly within the next year or two PPD-B may be generally available for use in assisting in differentiating between infection due to *M. tuberculosis* organisms and Group III Battey organisms by means of skin testing, that is perhaps where the reaction to PPD-S by standard Mantoux test is somewhere between 10 and 15 or 18 mm induration. A decision as to the causative organism could then be based on the relative size of reaction to both PPD-S and PPD-B. If the former were larger, this would indicate infection with *M. tuberculosis* organisms. If the latter were larger, then this would indicate infection with atypical Battey organisms.

Occasionally infection with *M. tuberculosis* organisms in children may not produce a very high level of hypersensitivity, particularly if the infection is recent and hypersensitivity is in the process of being elaborated, if the child has an overwhelming infection or if the hypersensitivity reaction is depressed from some other cause. For this reason and in an effort to try not to miss any cases where there may be clinical evidence of disease such as primary complex or progressive primary or miliary disease, clinical, radiological and/or bacteriological investigation and epidemiological follow-up should be carried out where the hypersensitivity is 5 mm or greater by standard Mantoux test. However, in tuberculosis control programs, priority should be given in those cases where the hypersensitivity is 10 mm or greater and such children should be considered for isoniazid treatment. Ideally, those with